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Vascular calcification in patients with preserved renal function

To the Editor: Vascular calcification was the subject of a recent review article [1], the subtitle of which was: Does preventing bone disease cause arterial disease? The authors discuss the possible link between calcium intake and cardiovascular disease. This link may be particularly important in patients with renal failure, but mention was made to the fact that a similar relation may exist in patients not on dialysis [2]. We have studied plasma calcium and phosphorus in 110 patients with coronary atherosclerosis and relatively preserved renal function (plasma creatinine smaller than 2 mg/dL) that underwent coronary angiography after an acute coronary syndrome [3]. Coronary artery disease burden was significant correlated with plasma calcium, but not with plasma phosphorus. A possible cut-off level for plasma calcium at 2.20 mmol/L was established [3]. These results are at odds with a previous report that showed that plasma phosphorus, but not plasma calcium, had an independent positive association with the angiographic severity of coronary disease, in a cohort of 376 stable patients without known renal disease [4]. It is unclear if subclinical renal failure (a condition in which phosphorus and calcium metabolism is changed) may have existed in this latter cohort. Plasma calcium [3] or plasma phosphorus [4] may be correlated with the severity of coronary atherosclerotic disease in patients with relatively preserved renal function [3], or in patients without known renal disease [4]. This may indicate a need for a reassessment of calcium supplementation, not only in dialysis patients, but also in patients with preserved renal function.

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Sevelamer-induced acidosis

To the Editor: Brezina et al [1] ignore an important but neglected aspect of gastrointestinal physiology in attributing sevelamer-induced acidosis to replacement of intestinal phosphate, bicarbonate, and bile acid anions by chloride carried on the resin. These three anions are minor luminal constituents in the distal large intestine, where sevelamer reaches its final equilibrium with intestinal fluids before expulsion from the body. The predominant anions here are short-chain fatty acid anions (SCFAA)—acetate, propionate, and n-butyrate, with total concentrations of over 150 mmol/L, constituting over 70% of luminal anions [2–4]. These SCFAA are derived from bacterial fermentation of food residues (mainly carbohydrate) and are bicarbonate precursors, normally being absorbed by the intestinal mucosa and incorporated into intermediary metabolism. Every mole of SCFAA removed from the body by sevelamer, and replaced by chloride from the resin, thus represents a loss of a mole of bicarbonate from the body and its replacement by chloride, equivalent to a gain by the body of a mole of hydrochloric acid; the amounts are far greater than would be generated by the resin's uptake of other anions from large bowel contents.

This uptake of SCFAA by an anion-exchange resin passing through the gut was shown over 40 years ago, when normal subjects were fed four different anion-exchange resins [5]. Recovered from stool, the resins' anion-exchange capacity was 66% to 84% unaccounted for by chloride, sulphate, phosphate, bicarbonate, and carbonate, and must have been SCFAA, which we were then unable to measure.

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